Right Precordial Lead (V₄R) ST-Segment Elevation Is Associated With Worse Prognosis in Patients With Acute Anterior Myocardial Infarction

To the Editor: The importance of ST-segment elevation (STE) in the right precordial leads during acute inferior myocardial infarction is well established. However, little is known about the significance of STE in the right precordial leads in patients presenting with acute anterior STE myocardial infarction (STEMI). In this study, we sought to examine the relationship between STE in right precordial lead V₄R during anterior STEMI and angiographic findings, wall motion abnormalities, and clinical outcomes.

We prospectively studied 117 consecutive patients admitted within 12 h of acute anterior STEMI. All patients underwent immediate coronary angiography with primary percutaneous intervention to the left anterior descending coronary artery (LAD). Early primary ventricular fibrillation (VF) was defined as VF occurring before coronary intervention. An episode of acute heart failure (HF) was recorded when a patient experienced signs and symptoms of HF and was prescribed intravenous diuretic agents during hospitalization. A complete 18-lead electrocardiogram (including 12 conventional leads, V₃R to V₅R, and V₇ to V₉) was recorded on admission. All STEs from the isoelectric line were measured manually 80 ms after the J point. Patients were stratified into 2 groups on the basis of the presence or absence of STE ≥1 mm in lead V₄R. The number of significantly diseased coronary arteries (>70% luminal diameter reduction) was determined.

Two-dimensional standard echocardiography was performed within 24 h of admission. Left ventricular regional function was assessed using a conventional 16-segment model. Each segment was assigned a score of 1 for normal, 2 for hypokinesis, 3 for akinesis, or 4 for dyskinesis. Wall motion score index was calculated as the mean score of all segments. To evaluate an association between specific segmental wall motion abnormality and STE in lead V₄R, we pre-specified 7 wall segments that are commonly involved in LAD-related STEMI: middle anterior, middle anteroseptal, middle septal, apical septal, apical anterior, apical lateral, and apical inferior.

Results are expressed as mean ± SD or as frequencies unless otherwise specified. Fisher exact, chi-square, Student t, and Mann-Whitney U tests were used as appropriate. To evaluate the association between right STE and the combined end point of primary VF, acute HF, and in-hospital death, we pre-specified 5 variables (age, sex, left ventricular ejection fraction, the sum of STE by echocardiography, and jeopardy score) and carried out logistic regression analysis adjusting for these covariates. In a secondary analysis, we evaluated the association between specific segmental wall motion abnormality and the presence of right STE; for this different end point, we pre-specified 7 cardiac wall segments and carried out logistic regression analysis adjusting for these 7 covariates only.

Of 117 consecutive patients with acute anterior STEMI, 39 (33%) had STE in lead V₄R and 78 (67%) did not. Both mean age (60 ± 11 years and 58 ± 14 years, respectively, p = 0.60) and the proportion of male patients (81% in both groups) were similar among patients with and those without STE in lead V₄R. Similarly, there were no statistically significant differences between the groups regarding cardiac risk factors, history of coronary events, and chronic medical treatment.

There were no significant differences between the groups regarding indexes of infarct size, including the sum of STE on conventional 12-lead electrocardiography (18 ± 8 mm and 19 ± 13 mm, p = 0.60), the number of leads with STE (6.1 ± 1.5 and 6.5 ± 2.2, p = 0.30), peak creatine kinase (median 2,323 IU/l [interquartile range: 627 to 3,452 IU/l] and 1,455 IU/l [interquartile range: 609 to 3,437 IU/l], p = 0.42), left ventricular ejection fraction by echocardiography (39 ± 11% and 40 ± 11%, p = 0.60), and wall motion score index (1.8 ± 0.5 and 1.7 ± 0.4, p = 0.40). None of the patients with STE in lead V₄R had echocardiographic evidence of right ventricular dysfunction or dilated right ventricles. Angiographic evaluation revealed no significant differences between the groups regarding initial and final Thrombolysis In Myocardial Infarction flow grade, proximal LAD involvement, and the number of significantly narrowed coronary arteries. After using logistic regression analysis using the 7 pre-specified LAD-related segments, only the middle anteroseptal segmental wall motion abnormality was significantly and independently associated with STE in lead V₄R. The odds ratio for akinesis (or more severe motion abnormality) was 6.1 (p = 0.036) and for hypokinesis (or more severe motion abnormality) was 12.0 (p = 0.033).

Patients presenting with compared those without STE in lead V₄R were more likely to experience the combined end point of primary VF, acute HF, or death (21 of 39 [54%] vs. 14 of 78 [18%], respectively, p < 0.001); they were also more likely to experience primary VF (8 of 39 [21%] vs. 2 of 78 [2.5%], respectively, p = 0.001) and acute HF (15 of 39 [39%] vs. 13 of 79 [17%], respectively, p = 0.009). In multivariate analysis, STE in lead V₄R on admission electrocardiography remained a strong independent variable associated with acute HF and the combined end point of primary VF, acute HF, or death during hospitalization (Table 1).

In the present study, we show that one-third of patients with anterior STEMI had STE in lead V₄R, which was associated with increased incidence of the combined end point of primary VF, acute HF, or death. Although STE in lead V₄R was not associated with larger infarct size, it was significantly associated with middle anteroseptal wall motion involvement. Thus, it seems that ischemia and infarction of a specific segment rather than infarct size...
account for the increase in both VF and acute HF risk in patients presenting with STE in lead V4R. The interventricular septum is known to play an important role in arrhythmias after myocardial infarction (1). Morita et al. (2) demonstrated that acute ischemia of the interventricular septum in canine cardiac wedge preparations produced asymmetric suppression of conduction velocity in the septum, and they suggested that such changes could contribute to the initiation of arrhythmia in patients with septal infarction. More recently, Sicouri et al. (3) demonstrated that dispersion of repolarization across the interventricular septum in canine wedge preparations is twice that of the left ventricular free wall, predisposing to development of Torsades de Pointes arrhythmias. The relatively low incidence of the combined end point (primary VF, acute HF, or both) calls for caution in the interpretation of the results, especially with regard to the multivariate analysis. Should our findings be confirmed in a larger cohort, we would recommend that right precordial leads be a routine part of the initial electrocardiographic study in all patients with anterior STEMI.

Alon Barsheshet, MD
Hanoch Hod, MD
Dan Oieru, MD
Ilan Goldenberg, MD
Amir Sandach, PhD
Roy Beigel, MD
Michael Glikson, MD
Micha S. Feinberg, MD
Michael Eldar, MD
*Shlomi Matetzky, MD

*Leviev Heart Center
Sheba Medical Center
Tel Hashomer 52621
Israel
E-mail: shlomi.matetzky@sheba.health.gov.il
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REFERENCES

Letters to the Editor

Proposed Diagnostic Criteria for Short QT Syndrome Are Badly Founded

I would like to commend Gollob et al. (1) for their attempt to generate criteria on how to diagnose short-QT syndrome (SQTS), but I would also like to point out some of the shortfalls and errors in their report.

In Table 1 of their report, at least 1 patient appears to have been included twice (Patients #23 and #49) with different ages and different QT intervals. Patient #46, published in 2008, was a 22-year-old man who “experienced unheralded syncope for the first time while driving, resulting in a motor vehicle accident” which in the current report is categorized as aborted cardiac arrest. This patient had no documented arrhythmias and no short QT interval on electrocardiography (QT interval 366 ms at 66 beats/min). The electrocardiogram appears to display early repolarization, and genetic testing discovered a novel KCNH2 mutation of undetermined significance, which was also present in the patient’s mother, who also had a normal QT interval. This patient does not fit the general concept of a patient with SQTS.

Patients #59 to #61, from a letter to the editor, are also outliers and should not have been included. These patients had a C-terminal KCNH2 mutation (R1135H) shown (2) to cause both a short QT interval and a Brugada-type electrocardiographic pattern, as seen in Patient #59.

The exclusion of Patients #59 to #61 would have changed Table 2 in their report. Instead of an upper value for the range of QT intervals in patients with SQTS of 401 ms, the actual value would have been 334 ms.

Using the QT interval corrected by Bazett’s formula in the proposed diagnostic criteria has important limitations. As already observed in the very first patient diagnosed with SQTS (3), the QT interval in SQTS changes very little with changes in heart rate, and correction of the QT interval is therefore barely necessary at normal